ISSN 2307-8960 (online)

World Journal of *Clinical Cases*

World J Clin Cases 2023 February 26; 11(6): 1224-1433

Published by Baishideng Publishing Group Inc

World Journal of Clinical Cases

Contents

Thrice Monthly Volume 11 Number 6 February 26, 2023

OPINION REVIEW

MINIREVIEWS

ORIGINAL ARTICLE

Retrospective Study

Clinical Trials Study

Observational Study

World Journal of Clinical Cases

Thrice Monthly Volume 11 Number 6 February 26, 2023

Contents

World Journal of Clinical Cases

Thrice Monthly Volume 11 Number 6 February 26, 2023

Contents

NAME OF JOURNAL
World Journal of Clinical Cases

ISSN
ISSN 2307-8960 (online)

LAUNCH DATE
April 16, 2013

FREQUENCY
Thrice Monthly

EDITORS-IN-CHIEF
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PUBLICATION DATE
February 26, 2023

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World Journal of Clinical Cases

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World J Clin Cases 2023 February 26; 11(6): 1410-1418

DOI: 10.12998/wjcc.v11.i6.1410

ISSN 2307-8960 (online)

CASE REPORT

Hepatitis A virus-associated acute acalculous cholecystitis in an adult-onset Still's disease patient: A case report and review of the literature

Chu-Heng Chang, You-Yang Wang, Yang Jiao

Specialty type: Medicine, general and internal

Provenance and peer review: Unsolicited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's scientific quality classification

Grade A (Excellent): 0
Grade B (Very good): B, B
Grade C (Good): 0
Grade D (Fair): 0
Grade E (Poor): 0

P-Reviewer: Ghannam WM, Egypt; Zarębska-Michaluk D, Poland

Received: November 11, 2022
Peer-review started: November 11, 2022
First decision: January 12, 2023
Revised: January 14, 2023
Accepted: February 8, 2023
Article in press: February 8, 2023
Published online: February 26, 2023

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Abstract

BACKGROUND

Acute acalculous cholecystitis (AAC) is inflammation of the gallbladder without evidence of calculi. Although rarely reported, its etiologies include hepatitis virus infection (*e.g.*, hepatitis A virus, HAV) and adult-onset Still's disease (AOSD). There are no reports of HAV-associated AAC in an AOSD patient.

CASE SUMMARY

Here we report a rare case of HAV infection-associated AAC in a 39-year-old woman who had a history of AOSD. The patient presented with an acute abdomen and hypotension. Elevated hepatobiliary enzymes and a thickened and distended gallbladder without gallstones on ultrasonography suggested AAC, but there were no signs of anemia nor thrombocytopenia. Serological screening revealed anti-HAV IgM antibodies. Steroid treatment did not alleviate her symptoms, and she was referred for laparoscopic cholecystectomy. The resected gallbladder was hydropic without perforation, and her clinical signs gradually improved after surgery.

CONCLUSION

AAC can be caused by HAV in AOSD patients. It is crucial to search for the underlying etiology for AAC, especially uncommon viral causes.

Key Words: Acalculous cholecystitis; Hepatitis A virus; Adult-onset Still's disease; Acute abdomen; Cholecystectomy; Case report

Core Tip: Acute acalculous cholecystitis (AAC) can be caused by hepatitis A virus (HAV) infection or adult-onset Still's disease (AOSD). Cholestasis is more likely to occur in HAV-associated AAC, whereas hematological complications are more common in AOSD-associated AAC. When AAC cannot be explained by AOSD, it is necessary to find other causes of AAC. An acute abdomen caused by HAV-related AAC requires careful consideration of the surgical necessity, since most cases are self-limiting and gallbladder perforation is rare.

Citation: Chang CH, Wang YY, Jiao Y. Hepatitis A virus-associated acute acalculous cholecystitis in an adult-onset Still's disease patient: A case report and review of the literature. *World J Clin Cases* 2023; 11(6): 1410-1418
URL: https://www.wjgnet.com/2307-8960/full/v11/i6/1410.htm
DOI: https://dx.doi.org/10.12998/wjcc.v11.i6.1410

INTRODUCTION

Acute acalculous cholecystitis (AAC) accounts for 2%-15% of all acute cholecystitis cases[1]. In contrast to acute calculous cholecystitis (ACC), no gallstones can be identified in the gallbladder in AAC, and its pathogenesis is thought to be related to ischemia-reperfusion injury after surgery or trauma, cholestasis caused by long-term fasting or intestinal obstruction, bacterial infection, or abnormal biliary tract anatomy[2-4]. Although rarely reported, hepatitis A virus (HAV) infection or adult-onset Still's disease (AOSD) can also cause AAC. HAV-associated AAC has mostly been reported in children and teenagers in developing countries, and it is often accompanied by fever, vomiting, transient liver dysfunction, and cholestasis[5,6]. On the other hand, AOSD is a chronic, systematic inflammatory disease characterized by recurrent fever, arthralgia, rash, and anemia or thrombocytopenia[7]. Patients with AOSD also may have high ferritin levels and sometimes concurrent AAC[8].

However, there are no reports of co-morbid AAC, HAV infection, and AOSD, which would represent a diagnostic and management challenge. Here we describe the case of a 39-year-old woman with AAC complicated by AOSD who was found to be anti-HAV IgM positive. We also searched the PubMed database with the keywords "[(hepatitis A virus) OR (adult-onset Still's disease)] AND acalculous cholecystitis" and found 14 HAV-associated AAC cases and three AOSD-associated AAC cases. Our case and review allow us to identify diagnostic clues that might help favor a particular diagnosis and discuss the necessity for surgical intervention to treat the AAC under these circumstances.

CASE PRESENTATION

Chief complaints

A 39-year-old woman presented to our hospital with a one-week history of fever (39-40 °C) and headache, chest tightness, and a sharp right upper quadrant pain.

History of present illness

The woman had a five-month history of AOSD, for which she had been taking oral methylprednisolone (26 mg/d) as maintenance therapy. She had suddenly developed a cough and sore throat two weeks previously, for which she was prescribed amoxicillin and clavulanate potassium (3.75 g/d), which was ineffective. In the week preceding admission, she had a fever of 39 °C accompanied by malaise, cutaneous icterus, and loss of appetite. Her methylprednisolone dose was increased to 48 mg/d and moxifloxacin hydrochloride (0.4 g iv, once) was administered, but this failed to control the symptoms.

History of past illness

The patient had ovarian endometriosis ten years previously and underwent laparoscopic ovarian cystectomy. She also had a history of a skin rash after taking a cephalosporin antibiotic.

Personal and family history

There was no personal nor family history of cholecystitis, nor was there a family history of AOSD or other auto-immune diseases.

Physical examination

On admission, she had cutaneous icterus and her temperature was 40 °C. Her blood pressure dropped to 83/45 mmHg and her heart rate increased to 100-110 bpm. Her liver was palpable under the costal margin, and she had abdominal distension and tenderness in the right upper quadrant and a positive Murphy's sign.

Laboratory examinations

Laboratory tests (Table 1) showed elevated lactate (1.9 mmol/L), white blood cell (WBC) count (17.34 × 10^9/L), and inflammatory markers [high-sensitivity C-reactive protein (hsCRP) 15.35 mg/L]. Moreover, total/direct bilirubin (TBil/DBil) (12.2/10.5 mg/dL) and hepatobiliary enzymes [aspartate aminotransferase (AST) 724 U/L, alanine aminotransferase (ALT) 223 U/L, gamma-glutamyl transferase (GGT) 735 U/L, alkaline phosphatase (ALP) 336 U/L] were elevated, and the prothrombin time (PT) was prolonged at 15.1 s. Her hemoglobin 14.7 g/dL and platelets 292 × 10^9/L were within normal limits. Further serological screening demonstrated anti-HAV IgM antibodies.

Imaging examinations

Ultrasonography revealed a dilated gallbladder (9.1 cm × 4.1 cm) with an evenly thickened wall (approximately 1.8 cm) and hepatosplenomegaly, but the intrahepatic bile duct was not dilated. Computed tomography (CT) suggested pericholecystic and hepatic fluid collection, a thickened gallbladder wall, a right pleural effusion, and ascites (Figure 1). No calculi were present.

FINAL DIAGNOSIS

The evidence suggested that the patient had HAV-associated AAC. The AAC could not be explained by the active state of AOSD, since steroid treatment did not alleviate the symptoms.

TREATMENT

Hypovolemia and septic shock were considered, and she was given supportive intravenous fluid treatment and norepinephrine as a vasoactive agent (0.2 μg/kg/min). However, the acute abdominal pain, chest tightness, and acute abdomen signs such as positive Murphy's sign and epigastric guarding continued. Gallbladder perforation was suspected, and she was referred for emergent laparoscopic cholecystectomy. During the surgery, the gallbladder was found to be hydropic without perforation with no evidence of calculi. Prednisone was maintained at the same dose (48 mg/d) after cholecystectomy.

OUTCOME AND FOLLOW-UP

Her clinical signs gradually improved after surgery. Microscopic examination of the gallbladder revealed normal epithelial architecture with mild lymphocyte infiltration. There was no perforation nor necrosis. During follow-up, her liver function returned to normal and the cutaneous icterus resolved.

DISCUSSION

Literature review

Here we present a case of AAC complicated by HAV infection and AOSD. We searched the PubMed database for published articles on the topic using the search terms "acalculous cholecystitis", "hepatitis A virus", and "adult-onset Still's disease". Fourteen patients have been reported in the literature with AAC due to acute HAV infection and three due to AOSD, and we compared these with our case.

Previous studies reporting HAV-associated AAC are summarized in Table 2. These patients commonly presented with fever (11/14), fatigue (7/14), nausea (5/14), vomiting (9/14), and abdominal pain (12/14). On physical examination, icterus (12/14), right upper abdominal tenderness (12/14), and an enlarged liver or spleen (6/12) were common. Ultrasonography and CT revealed thickened gallbladders accompanied by pericholecystic fluid and hepatosplenomegaly. Laboratory tests showed that all patients had elevated TBil/DBil, ALT, and AST. Anemia (2/8) and thrombocytopenia (3/10) occurred in several cases. However, despite advanced imaging and laboratory techniques, the diagnosis of complicated HAV-associated AAC as a cause of an acute abdomen seems to be challenging. Ciftci *et al* [10] presented a case of HAV-associated AAC in a child whose initial diagnosis was an acute abdomen due to blunt abdominal trauma. After physical examination, laboratory testing, and CT scanning, the patient was suspected to have gangrenous cholecystitis, but the exploratory laparotomy revealed no gallbladder necrosis nor perforation.

With respect to treatment and prognosis, most patients received conservative treatment (12/14) and only two patients underwent surgery. All patients had good outcomes. Most HAV-associated AAC cases were self-limiting, and the thickened, hydropic gallbladder decompressed within two weeks following conservative treatment. These findings were consistent with those of Kaya *et al*[18].

Table 1 Laboratory findings (at time of current admission)

Laboratory test	Laboratory value	Reference range
White blood cell count (WBC)	17.34	3.5-9.5 (× 10^9/L)
Hemoglobin (Hb)	14.70	11-15 (g/dL)
Platelets (PLT)	292.00	125-350 (× 10^9/L)
High-sensitivity C-reactive protein (hsCRP)	15.35	< 8.2 (mg/L)
Erythrocyte sedimentation rate (ESR)	7.00	0-20 (mm/h)
Aspartate aminotransferase (AST)	724.00	10-40 (U/L)
Alanine aminotransferase (ALT)	223.00	9-50 (U/L)
Gamma-glutamyl transferase (GGT)	735.00	8-55 (U/L)
Alkaline phosphatase (ALP)	336.00	40-100 (U/L)
Total bilirubin (TBil)	12.20	0.2-1.2 (mg/dL)
Direct bilirubin (DBil)	10.50	0-0.3 (mg/dL)
Creatinine (Cr)	58.00	45-84 (μmol/L)
Lactate	1.90	0.5-1.0 (mmol/L)
Prothrombin time (PT)	15.10	11-13 (s)
Activated partial thromboplastin time (APTT)	40.20	20-25 (s)

Cases of AAC in patients with AOSD (three cases) are summarized in Table 3. AOSD-associated AAC patients, all female, had recurrent fever, rash, and arthritis. On physical examination, two presented with a palpable liver and spleen, which was further confirmed by CT. All cases showed gallbladder enlargement or wall thickening but no calculi by ultrasonography or CT. Laboratory findings showed liver dysfunction (elevated TBil and hepatobiliary enzymes), anemia, and thrombocytopenia. Moreover, hyperferritinemia was presented in these patients, which might reflect the inflammatory state in autoimmune disease.

Arai *et al*[8] found several shared characteristics in AOSD patients. Two cases were complicated by macrophage activation syndrome (MAS) based on the findings of splenomegaly, cytopenia, and pathological changes in myeloid cells revealed by bone marrow biopsy. In addition, two cases were complicated by disseminated intravascular coagulation (DIC). The hypercytokinemia caused by MAS and widespread hypercoagulable state might aggravate multi-organ failure and severe illness. Furthermore, they showed that by inhibiting cytokine production and immune activation with glucocorticoids and cyclosporin A, both AAC and AOSD-related MAS or DIC could be resolved, suggesting that MAS and DIC might be secondary to their primary AOSD and that prompt and correct management of the primary disease can also slow or halt the progression of MAS and DIC.

It is worth considering whether surgery is needed in patients with AOSD-associated AAC. From the three previously reported cases, two patients received conservative treatment and one had cholecystectomy. The patient who underwent surgery[21] experienced hypovolemic shock, including no peripheral pulse and a systolic blood pressure of 50 mmHg. However, the surgery did not fully ameliorate the disease, since the patient experienced a rise in temperature after surgery and was later successfully treated with prednisone and naproxen. This leads us to consider whether surgical intervention is necessary in these cases. However, Arai *et al*[8] claimed that surgery should remain a treatment option for AOSD-associated AAC due to the possibility of gallbladder perforation as a complication. All three patients survived and had good outcomes. Overall, given the rarity of the condition, further reporting of individual cases would be helpful for guiding evidence-based treatment of AOSD-associated AAC.

Discussion

AOSD-associated AAC and HAV-associated AAC share several common characteristics. They both present with acute abdomen symptoms, elevated bilirubin and hepatobiliary enzymes, and imaging findings of hepatosplenomegaly and a thickened gallbladder without gallbladder calculi. However, cholestasis is often more severe in HAV-associated AAC, resulting in higher bilirubin levels and cutaneous icterus[22]. Meanwhile, hematological abnormalities are more obvious in AOSD-associated AAC: Anemia and thrombocytopenia were more frequent in AOSD-associated AAC, as was MAS or DIC[20]. In this patient, a differential diagnosis of MAS was considered. However, anemia, thrombocytopenia, and DIC were not present. Steroid treatment did not alleviate the patient's symptom, disfavoring AOSD as the cause of AAC in this case. Thus, based on serology and other laboratory

Table 2 Acute acalculous cholecystitis associated with hepatitis A virus infection

Ref.	Age/sex	Symptoms	Physical examination	Vital signs	Ultrasound/CT findings	Bilirubin (total/direct, mg/dL) (0.2-1.2/0-0.3)	AST/ALT (U/l) (10-40/9-50)	WBC (10^9/L) (3.5-9.5)	Hb (g/dL) (11-15)	Platelets (10^9/L) (125-350)
Mourani *et al* [9], 1994	68/M	Fever, chills, nausea, vomiting	Icterus, diaphoretic, hypotensive	/	Thickened gallbladder wall, acalculous	4.8/	5629/8670	/	/	/
Ciftci *et al* [10], 2001	7/M	Fever, fatigue, abdominal pain, mild respiratory distress	Icterus, abdominal distention, right upper quadrant tenderness	T 37.8 °C, HR 100 bpm, BP 100/70 mmHg	Subhepatic fluid, thickened gallbladder wall, acalculous	7.6/4.8	221/1288	8.8	13.9	/
Ozaras *et al* [11], 2003	28/M	Fatigue, abdominal pain, dark urine, anorexia, pale stool	Icterus, palpable liver with tenderness, murphy's sign (+)	/	Perihepatic fluid, thickened gallbladder wall, pericholecystic	8.4/3.9	370/1386	3.7	/	199
	20/F	Nausea, vomiting, fatigue, pruritus, and anorexia	Icterus, right abdominal tenderness, enlarged liver and spleen	/	Hepatosplenomegaly, hydropic gallbladder, acalculous	6.58/2.90	400/815	5.9	/	287
Basar *et al* [12], 2005	19/F	Fever, right upper abdominal pain	Icterus, right upper quadrant tenderness Murphy's sign (+)	/	Hepatomegaly, thickened gallbladder wall, acalculous, pericholecystic, intraabdominal fluid	11.6/5.7	984/1213	4.1	/	215
Bouyahia *et al*[13], 2008	14/M	Fever, vomiting, abdominal pain, myalgia	Right hypochondrium tenderness, enlarged liver and spleen	/	Thickened gallbladder wall, acalculous, pericholecystic fluid collection	4.97/3.33	1327/1112	5.2	13	130
Arroud *et al* [14], 2009	11/M	Fever, fatigue, vomiting, abdominal pain. myalgia, dark urine, pale stool	Icterus, enlarged liver and spleen	T 38.8 °C	Thickened gallbladder wall, acalculous, pericholecystic fluid collection	4.8/2.7	2953/1918	6.3	11.4	/
Suresh *et al* [15], 2009	2.5/F	Fever, fatigue nausea, vomiting, abdominal pain, loss of appetite, dark urine, pale stool	Icterus, tenderness in right side abdomen, Murphy's sign (+), enlarged liver and spleen	T 38.5 °C, HR 86 bpm, RR 22, BP 100/70 mmHg	Hepatosplenomegaly, thickened gallbladder wall, acalculous, perich-olecystic fluid	2.8/0.9	20.6/23.4	6.1	13.6	186
Souza *et al* [6], 2009	16/M	Fever, fatigue, nausea, vomiting, abdominal pain, cephalalgia	Diffuse abdominal pain to superficial and deep palpation	/	Hepatomegaly, thickened gallbladder wall, acalculous	5.01/3.69	1265/1046	/	14.2	112
Al-Amir *et al* [16], 2015	13/F	Fever, fatigue, vomiting, abdominal pain, dark urine, pale stool	Icterus, epigastric and right upper quadrant tenderness, Murphy's sign (+)	T 38.8 °C, other vital signs were normal	Thickened gallbladder wall, acalculous, pericholecystic fluid collection	16.1/12.3	3242/4298	4.5	15.4	/
Herek *et al* [5], 2011	9/M	Fever, nausea, vomiting, abdominal pain	Icterus, tenderness in the right upper quadrant, enlarged liver	T 37.9 °C, HR 84 bpm, BP 100/55 mmHg	Thickened gallbladder wall, acalculous, pericholecystic-free fluid	4.3/3.0	2261/2586	8.1	/	254
Prashanth *et al*[17], 2012	12/F	Abdominal pain and vomiting	Icterus, tenderness in the right hypochondrium	T 36.9 °C, HR 102 bpm, RR 18, BP 110/80 mmHg	Thickened gallbladder wall, acalculous	3.5/1.05	2150/2580	9	10	180

Kaya *et al* [18], 2013	31/F	Fever, nausea, abdominal pain, loss of appetite, back and joint pain, darkening of urine	Icterus, tenderness in the right side of the abdomen, Murphy's sign (+), enlarged liver	37.5 °C, HR 92 bpm, BP 110/60 mmHg	Hepatosplenomegaly, thickened gallbladder wall, acalculous, ascites	2.11/1.92	559/618	3.3	9.5	139
Aldaghi *et al* [19], 2015	5/M	Fever, abdominal pain	Icterus, mass in the right upper quadrant with tenderness	38 °C, HR 100 bpm, RR 30, BP 100/60 mmHg	Distended gallbladder, normal thickness, acalculous	5.3/3.9	516/722	8	/	426

HAV: Hepatitis A virus; AST: Aspartate transaminase; ALT: Alanine aminotransferase; BP: Blood pressure; RR: Respiratory rate; T: Temperature; WBC: White blood cell count; Hb: Hemoglobin; HR: Heart rate.

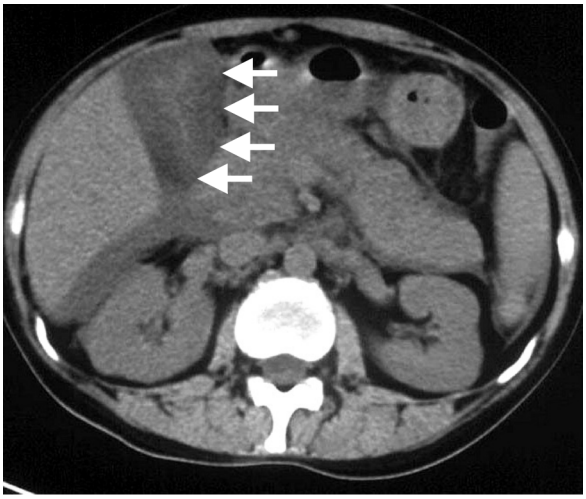
Table 3 Acute acalculous cholecystitis associated with adult-onset Still's disease

Ref.	Age/Sex	Symptoms	Physical examination	Vital signs	Ultrasound/CT findings	Bilirubin (total/direct, mg/dL) (0.2-1.2/0-0.3)	AST/ALT (U/l) (10-40/9-50)	WBC (10^9/L) (3.5-9.5)	Hb (g/dL) (11-15)	Platelets (10^9/L) (125-350)
Park *et al* [20], 2004	49/F	Recurrent fever, rash, and polyarthritis. Confused mental status on admission	Icterus, Murphy's sign (+), enlarged liver and spleen, facial rash, dehydrated tongue, equivocal neck stiffness, purpuras over the limbs, scabs of zoster	T 39 °C, BP 90/60 mmHg, HR 100 bpm, RR 30	Hepatosplenomegaly, thickened gallbladder wall, pericholecystic fluid collection, ileocolitis, right pleural effusion and ascites	3.7/2.8	453/154	7.1	9.5	17
Vallianou *et al*[21], 2014	28/F	Fever, vomiting, confused mental status, abdominal pain	Severe and diffuse abdominal tenderness	T 40 °C, no peripheral pulse, systolic BP 50 mmHg	Gallbladder enlargement with edema	/	/	/	/	/
Arai *et al* [8], 2021	21/F	Recurrent fever, rash, polyarthritis, nausea, vomiting, right hypochondriac pain	Rash, enlarged liver and spleen, tenderness in both shoulder and knee joints	T 40 °C	Hepatosplenomegaly, gallbladder enlargement and wall thickening, acalculous, cervical lymphadenopathy	2.15/1.56	763/469	2.3	11	79

AOSD: Adult-onset Still's disease; AST: Aspartate transaminase; ALT: Alanine aminotransferase; BP: Blood pressure; RR: Respiratory rate; T: Temperature; WBC: White blood cell count; Hb: Hemoglobin; HR: Heart rate.

findings, our final diagnosis for the patient was HAV-associated AAC.

Viral infections other than HAV may also lead to AAC. Hepatitis B virus[23,24] and hepatitis C virus [25,26] have both been reported as causes of AAC. Other viruses such as Epstein-Barr virus (EBV)[27,28], dengue virus[29,30], and human immunodeficiency virus[31,32] have also been implicated in AAC and have presented with an acute abdomen. More recently, coronavirus (COVID-19) has been reported in AAC cases[33], even leading to gangrenous cholecystitis[34]. Therefore, viral serology is an important diagnostic modality to search for a possible underlying etiology when a patient presents with AAC of unknown cause.



DOI: 10.12998/wjcc.v11.i6.1410

Figure 1 Computed tomography scan of the gallbladder and its surroundings. Axial computed tomography image confirmed distended gallbladder (9.1 cm × 4.1 cm) with an evenly thickened, hydropic gallbladder wall (approximately 1.8 cm). Pericholecystic and hepatic fluid was also seen. No calculi were present.

Due to the complexity of the case, our patient received intravenous fluid support, a vasoactive agent, steroid treatment, antibiotic management, and surgical intervention. Surprisingly, no perforation nor necrosis was found in the gallbladder after cholecystectomy. Due to the limitations of current imaging modalities and laboratory testing, it can be difficult to accurately determine the actual condition of the gallbladder prior to operation. However, as summarized previously, most HAV-associated AAC cases are self-limiting, and conservative management of AAC may be adequate[18]. Thus, cholecystectomy may be an option when faced with AAC but requires careful consideration and evaluation of the surgical necessity, not least given the positive outcomes of most patients with HAV-associated AAC with conservative therapy alone.

This study has several limitations. First, we only showed the association between hepatitis virus infection and AAC, and the cause-effect relationship between them is still debatable. Further validation of the cause of HAV-associated AAC requires evidence from animal experiments or cohort studies. Second, we did not examine whether the patient had hyperferritinemia, which is often present in active AOSD. However, our patient did not have anemia, thrombocytopenia, and did not develop DIC, and steroid treatment did not control the clinical course. These findings strongly disfavor active AOSD causing the AAC. This study is also limited by the availability of only three cases of AOSD-associated AAC, so we cannot be certain that these cases are representative. More cases of AOSD-associated AAC need to be described to verify our conclusions.

CONCLUSION

In conclusion, although AAC caused by HAV or AOSD is rare, it is possible that these conditions can overlap and complicate the diagnosis and management of AAC. When AAC cannot be explained by AOSD, it is important to search for other primary causes of AAC, and viral serology should form part of the diagnostic work-up. HAV-associated AAC is mostly self-limiting, and conservative therapy is usually adequate management for these patients unless gallbladder perforation is likely. Overall, however, the prognosis of AAC caused by HAV is very good, with conservative management the cornerstone of treatment.

FOOTNOTES

Author contributions: Jiao Y conceived this study; Chang CH and Wang YY drafted the manuscript; Jiao Y critically revised the manuscript; all authors have revised the final version of the manuscript and approved it for publication.

Supported by the National High Level Hospital Clinical Research Funding, No. 2022-PUMCH-A-017 and No. 2022-PUMCH-B-045; and CAMS Innovation Fund for Medical Sciences from Chinese Academy of Medical Sciences, No. 2021-I2M-1-062.

Informed consent statement: Informed written consent was obtained from the patient for publication of this report

and any accompanying images.

Conflict-of-interest statement: The authors declare that they have no conflict of interest.

CARE Checklist (2016) statement: The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016).

Country/Territory of origin: China

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S-Editor: Chen YL
L-Editor: A
P-Editor: Chen YL

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